

WEST Search History

Hide Items **Restore** **Clear** **Cancel**

DATE: Monday, February 02, 2004

Hide? Set Name Query Hit Count

DB=PGPB,USPT,JPAB,DWPI; PLUR=YES; OP=ADJ

<input type="checkbox"/>	L7	l1 same NFkB	1
<input type="checkbox"/>	L6	l4 and NFkB	1
<input type="checkbox"/>	L5	l4 and NFK	0
<input type="checkbox"/>	L4	TRADE alpha	3
<input type="checkbox"/>	L3	L1 and (NFK beta or NFK)	3
<input type="checkbox"/>	L2	L1 same (NFK beta or NFK)	0
<input type="checkbox"/>	L1	TRADE or TRADE alpha	173357

END OF SEARCH HISTORY

WEST Search History

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DATE: Monday, February 02, 2004

Hide?	Set Name	Query	Hit Count
		<i>DB=USPT,PGPB,JPAB,DWPI; PLUR=YES; OP=ADJ</i>	
<input type="checkbox"/>	L5	(TNF receptor member associated death protein) and @pd > 20021106	0
<input type="checkbox"/>	L4	(TRADE alpha or TRADE beta) and @pd > 20021106	0
<input type="checkbox"/>	L3	(L2 and (cell proliferation or cell death or apoptosis)) and @pd > 20021106	540
<input type="checkbox"/>	L2	(TRADE) and @pd > 20021106	29319
<input type="checkbox"/>	L1	(TNF Receptor member Associated with Death protein) and @pd > 20021106	0

END OF SEARCH HISTORY

factor receptor signaling pathway leading to ***NFkB*** activation.
AU Kuno, Kouji; Sukegawa, Kazuko; Ishikawa, Yuji; Orii, Tadao; Matsushima, Kouji (1)
CS (1) Dep. Pharmacol., Cancer Res. Inst., Kanazawa Univ., Takara-Machi 13-1, Kanazawa 920 Japan
SO International Immunology, (1994) Vol. 6, No. 8, pp. 1269-1272.
ISSN: 0953-8178.

DT Article

LA English

AB A recent report has suggested that tumor necrosis factor (TNF) utilizes acid sphingomyelinase (SMase) pathway to activate ***NFkB*** (Schulze et al. 1992. Cell 71:765). To directly investigate the role of acid SMase in IL-1 and ***TNF*** ***receptor*** -mediated signal transduction, we examined the ability of Niemann-Pick disease (NPD) type A fibroblasts, which are deficient in acid SMase, to induce IL-8 gene expression through activating ***NFkB***. Unexpectedly, IL-1-alpha and TNF-alpha efficiently induced IL-8 production and IL-8 mRNA in NPD type A fibroblasts as in normal fibroblasts. Furthermore, activation of ***NFkB*** was also induced in NPD type A fibroblasts in response to IL-1-alpha and TNF-alpha stimulation to a similar extent as in normal fibroblasts. These results provide evidence that acid SMase is not essential in IL-1 and ***TNF*** ***receptor*** signaling leading to ***NFkB*** activation as well as the cytokine gene activation which is regulated by ***NFkB***.

L17 ANSWER 29 OF 30 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92156130 EMBASE

DN 1992156130

TI Mechanisms of tumor necrosis factor action.

AU Schutze S.; Machleidt T.; Kronke M.

CS Inst. fur Med. Mikrobiologie/Hygiene, Technische Universitat Munchen, Trogerstr. 4a, 8000 Munchen 80, Germany

SO Seminars in Oncology, (1992) 19/2 SUPPL. 4 (16-24).

ISSN: 0093-7754 CODEN: SOLGAV

CY United States

DT Journal; Conference Article

FS 026 Immunology, Serology and Transplantation

029 Clinical Biochemistry

LA English

SL English

AB Tumor necrosis factor (TNF) is able to induce a great diversity of cellular responses via modulating the expression of a number of different genes. The multitude of TNF activities may be explained by both structural and functional heterogeneity in ***TNF*** ***receptors*** as well as by a diversification of postreceptor signal transduction pathways. Purification of ***TNF*** ***receptors*** has revealed two major, distinct binding proteins (TR60 and TR80). TR60 seems to be an essential component for TNF signaling; the functional role of TR80 remains to be elucidated. The pathway of postreceptor signal transduction involves phospholipase A2, a phosphatidylcholine-specific phospholipase C, protein kinase C, and other serine/threonine and tyrosine-specific protein kinases with as yet unknown function. At the receiving end of TNF signaling, induction of gene expression is mediated through activation of nuclear transcription factors, such as ***NFkB***, AP-1, IRF-1, and NF-GMa.

L17 ANSWER 30 OF 30 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 1991:38574 BIOSIS

DN BR40:15554

TI TNF INDUCED ***NFkB*** -HIV-1-LTR ACTIVATION CAN BE EFFICIENTLY BLOCKED

BY AN ANTI- ***TNF*** - ***RECEPTOR*** P60 ANTIBODY.

AU KRUPPA G; MEICHLE A; THOMA B; SCHEURICH P; PFIZENMAIER K; KROENKE M

CS CLINICAL RES. GROUP, MAX-PLANCK SOCIETY, GOSSLERSTR. 10D, 3400 GOETTINGEN, FRG.

SO SEVENTH INTERNATIONAL LYMPHOKINE WORKSHOP, SAN ANTONIO, TEXAS, USA,

OCTOBER 1-5, 1990. LYMPHOKINE RES. (1990) 9 (4), 573.

CODEN: LYREDH. ISSN: 0277-6766.

DT Conference

FS BR; OLD

LA English

=>

--Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
ENTRY	SESSION	
FULL ESTIMATED COST	168.93	268.60

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TOTAL ENTRY SESSION

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PASSWORD: TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 4 DEC 08 INPADOC: Legal Status data reloaded
NEWS 5 SEP 29 DISSABS now available on STN
NEWS 6 OCT 10 PCTFULL: Two new display fields added
NEWS 7 OCT 21 BIOSIS file reloaded and enhanced
NEWS 8 OCT 28 BIOSIS file segment of TOXCENTER reloaded and enhanced
NEWS 9 NOV 24 MSDS-CCOHS file reloaded
NEWS 10 DEC 08 CABA reloaded with left truncation
NEWS 11 DEC 08 IMS file names changed
NEWS 12 DEC 09 Experimental property data collected by CAS now available in REGISTRY
NEWS 13 DEC 09 STN Entry Date available for display in REGISTRY and CA/CAplus
NEWS 14 DEC 17 DGENE: Two new display fields added
NEWS 15 DEC 18 BIOTECHNO no longer updated
NEWS 16 DEC 19 CROPU no longer updated; subscriber discount no longer available
NEWS 17 DEC 22 Additional INPI reactions and pre-1907 documents added to CAS databases
NEWS 18 DEC 22 IFIPAT/IFIUDB/IFICDB reloaded with new data and search fields
NEWS 19 DEC 22 ABI-INFORM now available on STN
NEWS 20 JAN 27 Source of Registration (SR) information in REGISTRY updated and searchable
NEWS 21 JAN 27 A new search aid, the Company Name Thesaurus, available in CA/CAplus

NEWS EXPRESS DECEMBER 28 CURRENT WINDOWS VERSION IS V7.00, CURRENT

MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 23 SEPTEMBER 2003

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=> FIL BIOSIS EMBASE CAPLUS	SINCE FILE	TOTAL
COST IN U.S. DOLLARS	ENTRY	SESSION
FULL ESTIMATED COST	1.26	1.26

FILE 'BIOSIS' ENTERED AT 18:00:21 ON 02 FEB 2004
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=> s TRADE alpha
L1 3 TRADE ALPHA

=> s TRADE
L2 26507 TRADE

=> s l2 and NFK
L3 0 L2 AND NFK

=> s l2 and NFKB
L4 0 L2 AND NFKB

=> s l2 and NF-K
L5 0 L2 AND NF-K

=> s l2 and signaling pathway
L6 1 L2 AND SIGNALING PATHWAY

=> d bib abs

L6 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS
INC. on STN

AN 2002:245633 BIOSIS
DN PREV200200245633

TI Mechanisms of ageing: Public or private?

AU Partridge, Linda [Reprint author]; Gems, David

CS Department of Biology, University College London, Gower Street, London, WC1E 6BT, UK
l.partridge@ucl.ac.uk

SO Nature Reviews Genetics, (March, 2002) Vol. 3, No. 3, pp. 165-175. print.
ISSN: 1471-0056.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 17 Apr 2002

Last Updated on STN: 17 Apr 2002

=> dup rem l1

PROCESSING COMPLETED FOR L1

L7 3 DUP REM L1 (0 DUPLICATES REMOVED)

=> d bib abs 1-

YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L7 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2001:598038 CAPLUS

DN 135:175423

TI TRADE molecules and uses related thereto

IN Wood, Clive; Chaudhary, Divya; Long, Andrew

PA Genetics Institute, Inc., USA

SO PCT Int. Appl., 173 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2001058954 A2 20010816 WO 2001-US4238 20010209
WO 2001058954 A3 20020321
WO 2001058954 C2 20030116
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU,
ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

US 2002068696 A1 20020606 US 2001-780532 20010209

EP 1254176 A2 20021106 EP 2001-909036 20010209

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

JP 2004501604 T2 20040122 JP 2001-558100 20010209

PRAI US 2000-181922P P 20000211

US 2000-182148P P 20000214

WO 2001-US4238 W 20010209

AB The present invention relates, at least in part, to methods of modulating
proliferation and apoptotic state of cells using agents that modulate the
expression and/or activity of TRADE family polypeptides. In addn., the
invention provides two novel members of the TRADE family of mols.

L7 ANSWER 2 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS

INC. on STN

AN 2000:408685 BIOSIS

DN PREV200000408685

TI TRADE: A novel TNF-receptor family member.

AU Long, Andrew J. [Reprint author]; Bourque, Karen [Reprint author];
Chaudhary, Divya [Reprint author]; Haga, Hisanori; Tada, Hideaki; Burgess,
Paul [Reprint author]; Whitters, Matthew [Reprint author]; Tan, Xiang Yan
[Reprint author]; O'Hara, Denise [Reprint author]; Fitz, Lori [Reprint
author]; Beier, David; McCoy, John [Reprint author]; Collins, Mary
[Reprint author]; Shibayama, Shiro; Wood, Clive R. [Reprint author]

CS Genetics Institute, Inc., Wyeth Ayerst Research, Cambridge, MA, 02140, USA

SO Scandinavian Journal of Immunology, (June, 2000) Vol. 51, No. Supplement

1, pp. 64. print

Meeting Info.: 8th International TNF Congress, Conference on Tumor
Necrosis Factor and Related Molecules Scientific Advances and Medical
Applications. Trondheim, Norway. May 14-18, 2000.

CODEN: SJIMAX. ISSN: 0300-9475.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 27 Sep 2000

Last Updated on STN: 8 Jan 2002

L7 ANSWER 3 OF 3 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS

INC. on STN

AN 1993:206467 BIOSIS

DN PREV199395107692

TI Kinetic studies during enzyme hydrolysis of potato and cassava starches.

AU Gorinstein, Sheila

CS Dep. Pharmaceutical Chem., Sch. Pharmacy, Hebrew Univ. Jerusalem, Fac.
Med., P.O. Box 12065, Jerusalem 91120, Israel

SO Starch, (1993) Vol. 45, No. 3, pp. 91-95.

CODEN: STARDD. ISSN: 0038-9056.

DT Article

LA English

ED Entered STN: 23 Apr 1993

Last Updated on STN: 24 Apr 1993

AB The hydrolysis of raw potato and cassava starches by bacterial
alpha-amylase depends on the time of action, temperature and on the
specific starch involved. The molecular weight of the ***trade***
alpha -amylase (Termamyl 60L), determined by SDS-PAGE, was found

to

be 55-65 kDa. The properties of alpha-amylase such as kinetic parameters,
inhibition, stability, and thermostability were studied. The constants
K-m and maximum reaction rate V-max for alpha-amylase were fitted to
Michaelis-Menten models with these two starches. Differences in response
of potato and cassava starches to hydrolysis by Termamyl 60L can explain
differences found in K-m and V-max values and inhibition properties.

=> d his

(FILE 'HOME' ENTERED AT 17:56:59 ON 02 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:00:21 ON 02 FEB 2004

L1 3 S TRADE ALPHA

L2 26507 S TRADE

L3 0 S L2 AND NFK

L4 0 S L2 AND NFKB

L5 0 S L2 AND NF-K

L6 1 S L2 AND SIGNALING PATHWAY

L7 3 DUP REM L1 (0 DUPLICATES REMOVED)

=> s TNF-receptor

L8 7917 TNF-RECEPTOR

=> s l8 and trade

L9 3 L8 AND TRADE

=> dup rem l9

PROCESSING COMPLETED FOR L9

L10 3 DUP REM L9 (0 DUPLICATES REMOVED)

=> s l10 not l1

L11 1 L10 NOT L1

=> d bib abs

L11 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS

INC. on STN

AN 2000:377034 BIOSIS

DN PREV200000377034

TI ***TRADE*** , a novel ***TNF*** ***receptor*** superfamily

member, induces apoptosis and activates NFkappaB and Jnk.

AU Chaudhary, Divya [Reprint author]; Long, Andrew J. [Reprint author];
Bourque, Karen [Reprint author]; Adams, David H.; Hubscher, Stefan G.;
Towler, Paul [Reprint author]; Potts, Douglas [Reprint author]; Wood,
Clive R. [Reprint author]

CS Genetics Institute, Inc., Wyeth Ayerst Research, 87 Cambridge Park Drive,
Cambridge, MA, 02140, USA

SO Scandinavian Journal of Immunology, (June, 2000) Vol. 51, No. Supplement

1, pp. 33. print

Meeting Info.: 8th International TNF Congress, Conference on Tumor
Necrosis Factor and Related Molecules Scientific Advances and Medical
Applications. Trondheim, Norway. May 14-18, 2000.

CODEN: SJIMAX. ISSN: 0300-9475.

DT Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 6 Sep 2000

Last Updated on STN: 8 Jan 2002

=> s l8 and NFkappaB

L12 197 L8 AND NFKAPPAB

=> dup rem l12
PROCESSING COMPLETED FOR L12
L13 188 DUP REM L12 (9 DUPLICATES REMOVED)

=> d bib abs

L13 ANSWER 1 OF 188 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:879044 CAPLUS
DN 139:394859
TI Tumor Necrosis Factor Receptor-associated Factor 2 (TRAF2)-deficient B Lymphocytes Reveal Novel Roles for TRAF2 in CD40 Signaling
AU Hostager, Bruce S.; Haxhinasto, Sokol A.; Rowland, Sarah L.; Bishop, Gail A.
CS Interdisciplinary Program in Immunology, Department of Pediatrics, University of Iowa, Iowa City, IA, 52242, USA
SO Journal of Biological Chemistry (2003), 278(46), 45382-45390
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB CD40 function is initiated by tumor necrosis factor (***TNF***) ***receptor*** -assocd. factor (TRAF) adapter proteins, which play important roles in signaling by numerous receptors. Characterizing roles of individual TRAFs has been hampered by limitations of available exptl. models and the poor viability of most TRAF-deficient mice. Here, B cell lines made deficient in TRAF2 using a novel homologous recombination system reveal new roles for TRAF2. We demonstrate that TRAF2 participates in synergy between CD40 and B cell antigen receptor signals, and in CD40-mediated, TNF-dependent IgM prodn. We also find that TRAF2 participates in the degrdn. of TRAF3 assocd. with CD40 signaling, a role that may limit inhibitory actions of TRAF3. Finally, we show that TRAF2 and TRAF6 have overlapping functions in CD40-mediated NF- κ B activation and CD80 up-regulation. These findings demonstrate previously unappreciated roles for TRAF2 in signaling by ***TNF*** ***receptor*** family members, using an approach that facilitates the anal. of genes crit. to the viability of whole organisms.

RE.CNT 56 THERE ARE 56 CITED REFERENCES AVAILABLE FOR THIS RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l8 and NFkappaB and train
L14 0 L8 AND NFKAPPAB AND TRAIN

=> s l12 and train
L15 0 L12 AND TRAIN

=> s TRAIN-R
L16 3 TRAIN-R

=> dup rem l16
PROCESSING COMPLETED FOR L16
L17 2 DUP REM L16 (1 DUPLICATE REMOVED)

=> s l17 and l12
L18 0 L17 AND L12

=> d bib abs l17

L17 ANSWER 1 OF 2 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 1999106137 EMBASE
TI Primary nocturnal enuresis: Current.
AU Cendron M.
CS Dr. M. Cendron, Dartmouth-Hitchcock Medical Center, Lebanon, NH, United States
SO American Family Physician, (1 Mar 1999) 59/5 (1205-1214).
Refs: 42
ISSN: 0002-838X CODEN: AFPYAE
CY United States
DT Journal; Article
FS 007 Pediatrics and Pediatric Surgery
027 Biophysics, Bioengineering and Medical Instrumentation
028 Urology and Nephrology
037 Drug Literature Index
LA English
SL English
AB Primary nocturnal enuresis sometimes presents significant psychosocial problems for children and their parents. Causative factors may include maturational delay, genetic influence, difficulties in waking and decreased nighttime secretion of antidiuretic hormone. Anatomic abnormalities are usually not found, and psychologic causes are unlikely. Evaluation of enuresis usually requires no more than a complete history, a focused physical examination, and urine specific gravity and dipstick tests. Non-pharmacologic treatments include motivational therapy, behavioral conditioning and bladder-training exercises. Pharmacologic therapy includes imipramine, anticholinergic medication and desmopressin. These drugs have been used with varying degrees of success.

=> d bib abs l12 2

L12 ANSWER 2 OF 197 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS

INC. on STN
AN 2003:279222 BIOSIS
DN PREV200300279222
TI Endothelial activation by angiotensin II through ***NFkappaB*** and p38 pathways: Involvement of ***NFkappaB***-inducible kinase (NIK), free oxygen radicals, and selective inhibition by aspirin.
AU Costanzo, Antonio; Moretti, Francesca; Burgio, Vito Lelio; Bravi, Cristina; Guido, Francesco; Leviero, Massimo [Reprint Author]; Puri, Pier Lorenzo
CS Laboratory of Gene Expression, Fondazione A. Cesalpino, Universita' degli Studi di Roma "La Sapienza", Viale del Policlinico 155, 00161, Roma, Italy leviero@ifo.it; levmax@tin.it
SO Journal of Cellular Physiology, (June 2003) Vol. 195, No. 3, pp. 402-410. print.
CODEN: JCLLAX. ISSN: 0021-9541.
DT Article
LA English
ED Entered STN: 11 Jun 2003
Last Updated on STN: 11 Jun 2003
AB Angiotensin-II (All), the dominant effector of the renin-angiotensin system, is involved in the pathogenesis of cardiovascular diseases, such as atherosclerosis. Upregulation of the adhesion molecules VCAM-1, ICAM-1, and E-selectin in endothelial cells by inflammatory cytokines through nuclear factor kappa B (***NFkappaB***) activation is implicated in formation and progression of atherosclerotic plaque. Here we show that All induces ***NFkappaB*** -dependent transcription in primary endothelial cell lines, leading to the upregulation of ICAM-1 and VCAM-1 expression. ***NFkappaB*** activation by All is mediated by the ***NFkappaB*** -inducing kinase (NIK), a common mediator of ***NFkappaB*** activation by inflammatory cytokines, such as TNF-alpha. However, ***NFkappaB*** stimulation by All differs from that of TNF-alpha since a ***TNF*** - ***receptor*** associated factor 2 (TRAF-2) dominant negative mutant does not prevent All-mediated ***NFkappaB*** activation. In analogy with TNF-alpha-dependent activation of ***NFkappaB***, treatment with either the anti-oxidant N-acetyl cysteine (NAC) or the cyclooxygenase (COX) inhibitor acetyl salicylic acid (aspirin), but not indometacin, prevents the induction of ***NFkappaB*** -dependent transcription by All. Thus, production of reactive oxygen species, aspirin (asp)-sensitive enzymes of the arachidonate metabolism, and NIK are common transducers of All- and TNF-dependent pathways to ***NFkappaB***. All also activates the inflammatory p38 kinase in endothelial cells, an effect inhibited by exposure to either NAC or asp. Pharmacological interference of the p38 pathway, with the inhibitor SB 202190, prevented All-mediated activation of the ***NFkappaB*** target V-CAM, without affecting degradation of IkappaBalpha. These results support a pro-inflammatory effect of the vasoactive peptide All in endothelial cells, through at least two pathways- ***NFkappaB*** and p38-both of which are sensitive to asp and antioxidants.

=> d his

(FILE 'HOME' ENTERED AT 17:56:59 ON 02 FEB 2004)

FILE 'BIOSIS, EMBASE, CAPLUS' ENTERED AT 18:00:21 ON 02 FEB 2004
L1 3 S TRADE ALPHA
L2 26507 S TRADE
L3 0 S L2 AND NFK
L4 0 S L2 AND NFKB
L5 0 S L2 AND NF-K
L6 1 S L2 AND SIGNALING PATHWAY
L7 3 DUP REM L1 (0 DUPLICATES REMOVED)
L8 7917 S TNF-RECEPTOR
L9 3 S L8 AND TRADE
L10 3 DUP REM L9 (0 DUPLICATES REMOVED)
L11 1 S L10 NOT L1
L12 197 S L8 AND NFKAPPAB
L13 188 DUP REM L12 (9 DUPLICATES REMOVED)
L14 0 S L8 AND NFKAPPAB AND TRAIN
L15 0 S L12 AND TRAIN
L16 3 S TRAIN-R
L17 2 DUP REM L16 (1 DUPLICATE REMOVED)
L18 0 S L17 AND L12

=> s TNF receptor
L19 7917 TNF RECEPTOR

=> s l19 and r248
L20 1 L19 AND R248

=> d bib abs

L20 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN

AN 2001:397044 CAPLUS
DN 134:362290
TI Cloning of human ***TNF*** ***receptor*** ***R248*** cDNA and its use in the treatment of immune disorders
IN Kitson, Jeremy David Alistair
PA Glaxo Group Limited, UK

SO PCT Int. Appl., 34 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 2001038526 A1 20010531 WO 2000-GB4438 20001121
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR,
HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU,
SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,
YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY,
DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF,
BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI GB 1999-27681 A 19991123

AB A novel ***TNF*** ***receptor*** ***R248*** is provided which
is a screening target for the identification and development of novel
pharmaceutical agents which modulate the activity of the receptor and in
particular modulate activation of NF.kappa.B by the receptor. The present
invention provides the cDNA sequences coding for human ***TNF*** -
receptor ***R248*** polypeptide. A method for identification
of a substance that modulates ***TNF*** ***receptor*** activity
comprises contacting a polypeptide of the invention with a test substance
in the presence of a reporter whose activity is mediated by NFkB and
monitoring NFkB mediated activity.

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS
RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s TRAIN receptor
L21 1 TRAIN RECEPTOR

=> d bib abs

L21 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:194264 CAPLUS
DN 130:232854
TI TRAIN: A cysteine-rich member of the tumor necrosis factor receptor family
IN Tschopp, Jurg; Hession, Catherine
PA Biogen, Inc., USA
SO PCT Int. Appl., 32 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 2

PATENT NO. KIND DATE APPLICATION NO. DATE

PI WO 9913078 A1 19990318 WO 1998-US19030 19980911
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG,
KP, KR, KZ, LC, LK, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX,
NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT,
UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES,
FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI,
CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
CA 2301173 AA 19990318 CA 1998-2301173 19980911
AU 9892303 A1 19990329 AU 1998-92303 19980911
AU 738688 B2 20010927
EP 1012282 A1 20000628 EP 1998-944859 19980911
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, FI
JP 2002505843 T2 20020226 JP 2000-510863 19980911
US 2003219860 A1 20031127 US 2002-303502 20021122

PRAI US 1997-58631P P 19970912
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WO 1998-US19030 W 19980911
US 2000-522436 B1 20000309

AB A new member of the tumor necrosis factor receptor family, called TRAIN,
is identified and a cDNA encoding it is cloned and characterized. The
protein may be a target for use in treatment of tumors (no data).

RE.CNT 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS
RECORD

ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l1 and NFkappaB
L22 0 L1 AND NFKAPPAB

=>

---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	83.72	84.98

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE TOTAL
CA SUBSCRIBER PRICE	-2.77 -2.77

STN INTERNATIONAL LOGOFF AT 18:14:37 ON 02 FEB 2004

---Logging off of STN---

END

Unable to generate the STN prompt.
Exiting the script...